# Commentaries

## A Case Report of Lead Paint Poisoning during Renovation of a Victorian Farmhouse

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**Abstract:** We describe a series of four cases of childhood lead poisoning and two cases of adult lead toxicity in a professional family exposed to lead dust and fume during renovation of a rural farmhouse. Initial blood lead levels in the children ranged from 2.70 to 4.20 μM/L (56 to 87 μm/dl) and all four required chelation therapy. Lead-based paint poisoning, a well recognized entity among young children in poor, urban neighborhoods, is not confined exclusively to such areas. (*Am J Public Health* 1990; 80:1183–1185.)

*Introduction*

Lead-based paint has been recognized as a high-dose source of lead absorption and a cause of lead poisoning in young children since the beginning of the twentieth century.[1–7]

Epidemiologic studies of lead-based paint poisoning have tended to point the problem to housing units in urban areas, particularly in the northeastern and north central United States that received many coats of lead-based paint in the early years of the twentieth century.[8–10] Control of this problem began with passage in 1971 of the Lead-Based Paint Poisoning Prevention Act. Public health activities have been generally successful in reducing the incidence of acute lead-based paint poisoning.[11–13]

A newly recognized aspect of the problem is the occurrence of lead-based paint poisoning in children of middle- and upper-class families, occurring during the renovation or restoration of older houses. In urban areas the lead hazard has long been recognized, and there is usually some preparation to avoid the hazard. However, in suburban areas the population, up to now, has not been well informed. Families renovating old homes have often been unaware that the risk of lead poisoning pertained to them.[14–19]

We report here an outbreak of severe lead-based paint poisoning in a family exposed to lead dust and fume generated during removal of lead-based paint in a rural farm house.

*Case Reports*

Mr. and Mrs. A and their children, a five-year old daughter and a 20-month old son moved into their new home, a Victorian farm house in rural upstate New York in late June 1987.

In mid-October, the family noticed that their 10 year old mixed breed dog (Dog 1) began "shaking and twisting." She was brought to the family veterinarian who found her to be depressed, dehydrated, weak, and to have an abscessed molar. She was hydrated with intravenous fluids, had her molar extracted, and was started on antibiotic therapy. Laboratory values of note included increased nucleated red blood cells without anemia, elevated white blood count 26.6 $10^6$/L(26,600/cumm), mildly elevated serum glucose (126 mg/dL) and isosthenuric urine with 2+ protein, 0.25% glucose and 2+ granular casts.

The veterinarian suspected lead poisoning. He questioned Mrs. A about sources of exposure to lead. She explained that their home had been undergoing extensive renovation. Both family dogs had been at home throughout the renovation process. Dog 1, in particular, had been noted to have a fondness for one of the workmen and sat by his feet while he sanded old paint. She was often seen licking her dusty coat.

A blood sample for lead determination was drawn from Dog 1. The dog was then started on a course of calcium disodium EDTA, 400 mg subcutaneously, three times daily for two days, followed by a course of cupramine 250 mg orally three times per day. Unfortunately, the pre-chelation blood sample was unacceptable to the laboratory; a second sample was therefore sent for analysis after the second day of chelation therapy. The blood lead level was 2.46 μM/L (51 μgm/dl) (usual range 5 to 25 μm/dL). The dog improved and was sent home to complete her 10-day course of cupramine therapy. However, after three days, she developed weakness and anorexia and died shortly thereafter due to renal failure.

Dog 2, a 10-year old Samoyed was found to have a blood level of 3.38 μM/L. She was treated with a 10-day course of cupramine therapy and has done well.

Mrs. A reported in November that she had been feeling tired and weak. Her daughter complained of stomach aches on several mornings prior to boarding the school bus. Mr. A had an episode of severe nausea after spending a weekend at home while renovation work, including the use of torches, was proceeding.

A free erythrocyte protoporphyrin level was obtained for Mrs. A and found to be 130 μM/L. Her 20-month old son (child 1) had an FEP of 281 μM/L and her five-year old daughter (child 2) had an FEP of 149 μM/L. Mrs. A and her daughter were given intramuscular calcium disodium EDTA.

One week later, Mrs. A was found to have a blood lead

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level of 2.27 μM/L. Her daughter's lead level at that time was 2.70 μM/L, and her son's 4.20 μM/L. All three were admitted to a local hospital for chelation therapy with calcium disodium EDTA. Mr. A was found at this time to have a blood lead level of 1.59 μM/L.

A repeat blood lead level performed on Mrs. A six weeks after a five-day course of calcium disodium EDTA chelation therapy was 1.30 μM/L. Although improved, she was still feeling rather weak and tired. A pregnancy test was performed two weeks after discharge. She was found to be eight weeks pregnant and opted for therapeutic abortion.

Both children were initially chelated for five days, given a two-day rest and chelated again. Blood lead levels two weeks after the second course of chelation were 2.61 μM/L for the 20-month old boy (child 1) and 1.83 μM/L for the five-year old girl (child 2). Both children required subsequent chelation therapy. Only one additional course was required for child 2. However, child 1 required three additional courses of chelation therapy. Follow-up lead level three weeks post discharge from the fourth course was 1.59 μM/L with a free erythrocyte protoporphyrin level of 626 μM/L. A fifth course of chelation took place about six weeks later. Close follow-up continues.

Two months after completion of the repair work, Mr. A had a blood lead level of 1.93 μM/L and a free erythrocyte protoporphyrin of 150 μM/L. He has been entirely asymptomatic.

The children's baby-sitter, a 24-year old woman who cared for the children in their home, was found during the second week of November to have a blood lead level of 0.87 μM/L. Her children, a two-year old daughter (child 3) and a three-year old son (child 4), were also tested. Child 3 had a blood lead level of 3.23 μM/L and child 4 had a level of 2.75 μM/L. The children were admitted to hospital and underwent a five-day course of chelation therapy with calcium disodium EDTA. Blood lead levels two weeks post chelation therapy were 0.92 μM/L for child 3 and 0.72 μM/L for child 4.

Unfortunately there is no information available concerning the two workers who performed the renovation work and presumably had the heaviest exposure.

*Environmental Background*

The family home is a two story, wood and stone nineteenth century farmhouse with 10 rooms arranged around a center hallway with a center staircase. Most of its solid wooden floors, wooden moldings, and door frames had been covered with multiple coats of lead-based paint. The walls had been covered with multiple coats of lead-based paint and wall paper.

In a 10-week period, from mid-August through the last week in October of 1987, the house underwent major renovations, including restoration of the floor, doorways, and wooden moldings. This work involved removing the multiple coats of paint down to the original wood, which was then coated with varnish. Some of the walls were stripped of paint and paper, and two ceilings required extensive repair. Two workmen used rotary and hand sanders to remove old paint and paper from the walls and floors. They used torch, heat gun, and chemical wood stripper to remove paint from the moldings and door frames.

To avoid the renovation, the family left the house for vacation in early August and returned in mid-September. Upon their return, the family found the work to be only partially completed. Work areas had not been sealed off while the work was being performed. A thick dust had formed and spread throughout the house. Partial clean up had been performed immediately prior to the family's return.

The work continued after the family returned from vacation and went on until late October. In this period the doorways of the central hallway were stripped of their paint by torch. Some of the moldings were taken into the front yard and torched by Mrs. A but most were stripped in place by the workmen.

During this period the family tried to stay out of the work area. This was not difficult for Mr. A who worked in New York City and traveled on business. Mrs. A, on the other hand, worked in a home office and cared for their children at home. Child 2 attended kindergarten five days a week for half a day. During the last week of September, in order to help ensure the children's safety, the baby sitter was hired to keep the children occupied (usually playing outside of the house). She was accompanied by her two children, a girl age two (child 3) and a boy age three (child 4). For three to four weeks, she worked at the home five hours a day, five days a week. For the remaining two weeks she worked only two days per week.

Subsequent to the clinical diagnosis of lead poisoning, the county health department has evaluated the walls, ceilings, and moldings of the rooms by x-ray diffraction. This testing revealed markedly elevated levels of lead in the painted areas that had not undergone renovation. Most levels were greater than 10.0 milligrams of lead per square centimeter of surface area; the acceptable level is considered to be less than 0.7 milligrams of lead per square centimeter.

*Discussion*

Many old homes are undergoing restoration today by new owners. Sanding, torching, and the use of heat guns to remove the old layers of paint are common. These methods produce chips, fine dusts, and fumes that can be ingested or inhaled. Lead fumes which are generated by heat removal, using either torches or blowers, are especially dangerous, because they consist of small, easily respirable particles, less than one micron in diameter; exposure to lead fumes can produce acute lead poisoning within days or even hours.[20] Lead dusts can invade all areas of a home, making the cleaning process very difficult and producing continued exposure.[21–23] Also, the resulting chips may be eaten by small children and animals. Lead poisoning can result.

Unfortunately, most families outside of urban centers are unaware of the potential dangers of lead poisoning or the precautions necessary to prevent lead-based paint poisoning.[24–27] It is extremely important that parents and practitioners be aware of the hazards of lead, its sources and routes of absorption, and about safe approaches to the prevention of exposure. It is equally essential that once potential sources of exposure are discovered, that everyone, especially children and pregnant women, are removed from exposure until adequate clean up had been performed.

The present cases illustrate that illness in a family pet can, in fact, be the sentinel event in a toxic environmental exposure. This emphasizes the need for good communication between primary care physicians and veterinary medicine practitioners.[15,27–29]

Childhood lead poisoning remains widespread. Between 1976 and 1980, 4 percent of children ages 1 to 6 years were found in the Second National Health and Nutrition Survey (NHANESII) to have blood lead concentrations of 1.45

μM/L or more. The present definition of childhood lead poisoning is a whole blood lead concentration of 1.21 μM/L (30 μgm/dl) with an erythrocyte protoporphyrin level of 65 μM/L. Prevention of lead poisoning in young children requires a high index of suspicion and widespread screening of young children in urban, suburban, and rural areas.[26]

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